

Epitomes

Important Advances in Clinical Medicine

Dermatology

The Scientific Board of the California Medical Association presents the following inventory of items of progress in dermatology. Each item, in the judgment of a panel of knowledgeable physicians, has recently become reasonably firmly established, both as to scientific fact and important clinical significance. The items are presented in simple epitome and an authoritative reference, both to the item itself and to the subject as a whole, is generally given for those who may be unfamiliar with a particular item. The purpose is to assist busy practitioners, students, research workers or scholars to stay abreast of these items of progress in dermatology that have recently achieved a substantial degree of authoritative acceptance, whether in their own field of special interest or another.

The items of progress listed below were selected by the Advisory Panel to the Section on Dermatology of the California Medical Association and the summaries were prepared under its direction.

Reprint requests to Division of Scientific and Educational Activities,
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Sunscreens

THE SUNBURN effects of ultraviolet light on epidermal DNA are well documented, and whereas a definite cause and effect relation between such damage and skin carcinogenesis is not certain, DNA damage induced by ultraviolet-B is thought to be an important component. An ideal sunscreen should protect against all biologic effects of ultraviolet radiation and not simply inhibit ultraviolet-induced skin erythema.

There have been recent attempts to evaluate sunscreens using different assay procedures. The standard human assay procedure is called the human sun protection factor (SPF) assay. Using volunteers, the minimal erythema dose is determined in their skin where not previously exposed to sun by administering a series of dose increments of ultraviolet radiation using usually a solar-simulator light source. The next day, areas of skin are treated with the sunscreen to be tested and the ultraviolet radiation again delivered. The sun protection factor is calculated as the ratio of the minimal erythema dose in protected skin to the minimal erythema dose in unprotected skin.

Many commercially available sunscreens have sun protection factor readings of 15; this means that a person can stay in the sun for 15 times the length of time that would produce skin erythema without the sunscreen.

Sunscreens containing para-aminobenzoic acid (PABA) and its esters have been shown to protect against ultraviolet-induced changes in epidermal DNA synthesis rates in hairless mice. They have also been shown in hairless mice to protect against photocarcinogenesis.

Whereas definitive information on the ability of sun-

screens to protect against photocarcinogenesis in humans is not yet available, logic tells us that these sunscreen preparations are highly likely to inhibit photocarcinogenesis and possibly photoenhanced aging in human skin.

The recommendation must be that all persons, particularly those with light skin, should use sunscreens routinely and regularly when spending time exposed to the sun.

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Kligman LH, Akin FJ, Kligman AM: Sunscreens prevent ultraviolet photocarcinogenesis. *J Am Acad Dermatol* 1980 Jul; 3:30-35
Lowe NJ, Breeding J: Evaluation of sunscreen protection by measurement of epidermal DNA synthesis. *J Invest Dermatol* 1980 Mar; 74:181-182
Sayre RM, Marlowe E, Agin PP, et al: Performance of six sunscreen formulations on human skin: A comparison. *Arch Dermatol* 1979 Jan; 115:46-49

Acyclovir and Herpes Simplex

ACYCLOVIR (acycloguanosine; 9-[(2-hydroxyethoxy)methyl]guanine; Zovirax, Burroughs Wellcome Co.) is emerging as but one of a new generation of specific antiviral chemotherapeutic agents that are potent inhibitors of herpesvirus, the organisms that cause herpes simplex types 1 and 2, herpes zoster and varicella. The selectivity of the drug is due to the fact that cells harboring the herpesvirus contain thymidine kinase, an enzyme that transforms acyclovir into acycloguanosine triphosphate. Uninfected cells lack thymidine kinase and acycloguanosine triphosphate, a potent inhibitor of viral DNA polymerase, forms in herpes-infected cells at 40 to 100 times greater concentration than uninfected cells. The drug has a low index of toxicity for humans. Acyclovir has been licensed by the Food and Drug Administration in both topical and intravenous forms for treatment of a first episode of herpes simplex in both healthy and immunocompromised patients.